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ProductInformation

Anti-SMC1L1
produced in rabbit, IgG fraction of antiserum

Catalog Number **S6821**

Product Description

Anti-SMC1L1 is developed in rabbit using as immunogen a synthetic peptide corresponding to amino acids 954-970 of human SMC1L1, conjugated to KLH via an N-terminal cysteine residue. This sequence differs from the corresponding mouse and rat sequences in 2 and 3 amino acids, respectively. Whole antiserum is fractionated and then further purified by ion-exchange chromatography to provide the IgG fraction of antiserum that is essentially free of other rabbit serum proteins.

Anti-SMC1L1 (also known as SMC1 and SMC1α) specifically recognizes SMC1L1 by immunoblotting (160 kDa) and immunoprecipitation. Staining of the SMC1L1 band in immunoblotting is specifically inhibited by the immunizing peptide.

Proper cohesion of sister chromatids is a prerequisite for the correct segregation of chromosomes during cell division. The cohesin chromosome complex is required for sister chromatid cohesion.[1] This complex is partly composed of two structural maintenance of chromosomes (SMC) proteins, SMC3, and either SMC1L1 (SMC1, SMC1α) or SMC1L2. The diverse functions of the SMC1 proteins range far beyond chromosome segregation and involve nearly all aspects of chromosome behavior including DNA repair.[1-3] SMC1 and SMC2 were originally identified in *Saccharomyces cerevisiae* as genes required for proper condensation and segregation of chromosomes.[4] There are at least six SMC family members that form three heterodimers in specific combinations: SMC1L1 (SMC1) and SMC3 constitute the core of the cohesin complex that maintains sister chromatid cohesion, whereas SMC2 and SMC4 are components of the condensin complexes that mediate chromosome condensation during mitosis. A third complex, containing SMC5 and SCM6, is mainly involved in the cellular response to DNA damage in yeast as well as humans.[1, 5, 6] Although SMC1L1 was originally identified as a component of cohesin, it has been shown to be phosphorylated by ATM indicating a potential role for this protein in DNA repair. ATM (ataxia telangiectasia mutated) and ATR (ataxia telangiectasia and Rad3 related) are key signal transducers of the DNA damage response. SMC1L1 is phosphorylated at Ser^{957} and Ser^{966} in an ATM-dependent manner after ionizing irradiation in response to DNA damage.[7, 8] ATM appears to be the initiating event in cells following DNA-strand breakage with the magnitude of this activation being influenced by NBS1.[9] Both NBS1 (Nibrin) and BRCA1 (Breast cancer onset 1) migrate then to sites of DNA breaks and recruit activated ATM, which in turn phosphorylates SMC1L1.[9] Thus, SMC1L1 phosphorylation appears to be the critical downstream event in the ATM-NBS1-BRCA1 pathway, which mediates cell survival and chromosomal stabilization after DNA damage.[10, 11] Interestingly, mutations in SMC1L1 were found in the Cornelia de Lange syndrome, a multisystem developmental disorder.[12]

Reagent

Supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide.

Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

For continuous use, store at 2-8 °C for up to one month. For extended storage, freeze in working aliquots. Repeated freezing and thawing, or storage in "frost-free" freezers, is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilutions should be discarded if not used within 12 hours.

Product Profile

Immunoblotting: a working dilution of 1:2.000-1:4,000 is recommended using extracts of HeLa nuclear cells.

Immunoprecipitation: 5-10 µL of the antibody immunoprecipitates SMC1L1 from 293-T cell lysates.

Note: In order to obtain the best results using various techniques and preparations, we recommend determining the optimal working dilutions by titration**.**

References

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